Chapter 7
Risk and Hazard Characterization

What's Covered in Chapter 7:

- Individual Risk and Hazard Estimation
- Quantitative Estimation of Cancer Risk
- Quantitative Estimation of Noncancer Effects
- Target Levels
- Acute Exposure Resulting from Direct Inhalation

Risk characterization must exhibit the core values of transparency, clarity, consistency, and reasonableness. The final step of a risk assessment is the calculation of the upper-bound excess lifetime cancer risks (risk) and noncarcinogenic hazards (hazard) for each of the pathways and receptors identified in Chapter 4. Risks and hazards are then summed for specific receptors, across all applicable exposure pathways, to obtain an estimate of total individual risk and hazard for specific receptors.

Risk from exposure to combustion emissions is the probability that a receptor will develop cancer, based on a unique set of exposure, model, and toxicity assumptions. The slope factor is used in risk assessments to estimate and upper bound lifetime probability of an individual developing cancer as a result of exposure to a particular level of a potential carcinogen. For example, a risk of 1×10^{-5} is interpreted to mean that an individual has no more than, and likely less than, a one in 100,000 chance of developing cancer from the exposure being evaluated. In contrast, hazard is quantified as the potential for developing noncarcinogenic health effects as a result of exposure to COPCs, averaged over an exposure period. A hazard is not a probability but, rather, a measure (calculated as a ratio) of the magnitude of a receptor's potential exposure relative to a standard exposure level (*RfD* or *RfC*). The standard exposure level is calculated over a similar exposure period and is estimated to pose no appreciable likelihood of adverse health effects to potential receptors, including special populations (U.S. EPA 1989e). Risks and hazards are typically characterized for a single receptor and are referred to as individual risks and hazards (U.S. EPA 1989e; 1994g; NC DEHNR 1997).

At least one U.S. EPA guidance document, concerning the characterization of risks and hazards associated with combustion facilities, suggests that population risks and hazards should be calculated in addition to individual risks (U.S. EPA 1993h). Population risk is defined as the aggregate risk of the exposed population; it takes into account the risk associated with various exposure scenarios and the number of individuals represented by each exposure scenario. Therefore, U.S. EPA OSW recommends that the risk assessment address only the individual risks and hazards; calculation of population risks and hazards is not required. However, if a permitting authority feels that site-specific conditions indicate calculation of population risks should be considered, U.S. EPA OSW recommends following the methodologies described in U.S. EPA NCEA document, *Methodology for Assessing Health Risks Associated with Multiple Exposure Pathways to Combustor Emissions* (In Press).

INFORMATION RECOMMENDED FOR RISK ASSESSMENT REPORT

- Indicate the scope of the risk assessment (match the level of effort to the scope)
- Summarize the major risk conclusions.
- Identify key issues (a key issue is critical to properly evaluate the conclusions). For example, was surrogate or measured emissions data used.
- Describe clearly the methods used to determine risk (provide qualitative narration of the quantitative results).
- Summarize the overall strengths and major uncertainties.

7.1 ESTIMATION OF INDIVIDUAL RISK AND HAZARD

Individual risk and hazard descriptors are intended to convey information about the potential risks to individuals potentially impacted by emissions from a facility burning hazardous waste. A risk assessment developed by following the procedures described in Chapters 2 through 6 and Appendixes B and C will provide (1) quantitative and qualitative estimates of risk and hazard associated with exposure to COPCs, (2) estimates of health effects associated with exposure to lead, (3) evaluation of infant

exposure to 2,3,7,8-TCDD TEQ present in breast milk, and (4) evaluation of acute exposure resulting from direct inhalation.

7.2 QUANTITATIVE ESTIMATION OF CANCER RISK

As described above, for carcinogenic chemicals, risk estimates represent the incremental probability that an individual will develop cancer over a lifetime as a result of a specific exposure to a carcinogenic chemical (U.S. EPA 1989e). These risks are calculated as follows:

$$Cancer\ Risk = LADD \cdot CSF \qquad \text{Equation 7-1}$$

where

LADD = Lifetime average daily dose (mg/kg-day)
CSF = Cancer slope factor $(mg/kg\text{-}day)^{-1}$

Within a specific exposure pathway, receptors may be exposed to more than one COPC. The total risk associated with exposure to all COPCs through a single exposure pathway is estimated as follows (U.S. EPA 1989e):

$$Cancer\ Risk_T = \sum_i Cancer\ Risk_i \qquad \text{Equation 7-2}$$

where

$Cancer\ Risk_T$ = Total cancer risk for a specific exposure pathway
$Cancer\ Risk_i$ = Cancer risk for COPC *i* for a specific exposure pathway

At particular exposure scenario locations, receptors may be exposed through a number of exposure pathways (see Table 4-1). Risks from multiple exposure pathways should be summed for a given receptor specific to each recommended exposure scenario. That is, risks should be summed across the receptor-exposure pathway combinations, which are identified in Table 4-1. In the context of risk assessments which evaluate the emissions from hazardous waste combustion units, the risks from all RCRA regulated combustion units that are permitted, have interim status, or expected to be constructed, should be summed for each receptor. For fugitive emissions from storage and handling of hazardous, the risk associated with fugitive emissions should be added to the risks from the combustion unit for each receptor at each exposure scenario location. For example, if a facility operates both an incinerator and a

boiler that burn hazardous waste, then the risks from both types of units should be summed across all the units for each receptor. The total risk posed to a receptor is the sum of total risks from each individual exposure pathway expressed as follows:

$$Total\ Cancer\ Risk = \sum CancerRisk_T \qquad \text{Equation 7-3}$$

where

Total Cancer Risk	=	Total cancer risk from multiple exposure pathways
$Cancer\ Risk_T$	=	Total cancer risk for a specific exposure pathway

Equations used to calculate dose and risk levels are presented in Appendix C. Appendix A-3 presents oral and inhalation slope factors (*CSF*) for many potential COPCs. However, for each risk assessment, the IRIS and HEAST databases should be checked for updated values. If toxicity values for COPCs not identified in Appendix A-3 are included in the risk assessment, *CSF*s for these compounds can be obtained from the following sources, listed in the preferred order: (1) U.S. EPA's IRIS (U.S. EPA 1996a) and (2) U.S. EPA HEAST (U.S. EPA 1994b).

In the assessment of carcinogenic risk from COPCs, U.S. EPA-derived or reviewed health benchmarks (*CSFs*, *URFs*, and *Inhalation CSFs*) are recommended. However, for numerous compounds, a complete set of inhalation and oral EPA-derived health benchmarks are not available. In such cases, the health benchmarks presented in Appendix A-3 were calculated based on available U.S. EPA-derived benchmarks values.

If relevant information is not available from these sources, the applicant should contact the appropriate permitting authority, which may be able to assist in developing the necessary toxicity values. For example, Minimum Risk Values published by the Agency for Toxic Substances and Disease Registry (ASTDR) may be used at the discretion of the permitting authority.

7.3 QUANTITATIVE ESTIMATION OF POTENTIAL FOR NONCANCER EFFECTS

Standard risk assessment models assume that noncarcinogenic effects, exhibit a threshold; that is, there is a level of exposure below which no adverse effects will be observed (U.S. EPA 1989e). The potential for

noncarcinogenic health effects resulting from exposure to a chemical is generally assessed by (1) comparing an exposure estimate (see Chapter 6) to an *RfD* for oral exposures, and (2) comparing an estimated chemical-specific air concentration to the *RfC* for direct inhalation exposures. An *RfD* is a daily oral intake rate that is estimated to pose no appreciable risk of adverse health effects, even to sensitive populations, over a specific exposure duration. Similarly, an *RfC* is an estimated daily concentration of a chemical in air, the exposure to which over a specific exposure duration poses no appreciable risk of adverse health effects, even to sensitive populations (U.S. EPA 1989e).

The exposure durations assumed for the exposure pathways identified in Table 4-1 range from subchronic to chronic in relative length. However, chronic *RfDs* and *RfCs* should be used to evaluate all exposure pathways. In the absence of a chronic *RfD*, a subchronic *RfD* with an Uncertainty Factor (3 to 10) can be considered. The comparisons of exposure estimates and COPC-specific air concentrations to *RfD* and *RfC* values, described above, are known as hazard quotients (*HQ*), which are calculated as follows:

$$HQ = \frac{ADD}{RfD} \quad \text{or} \quad HQ = \frac{C_a}{RfC} \qquad \text{Equation 7-4}$$

where

HQ	=	Hazard quotient (unitless)
ADD	=	Average daily dose (mg/kg-day)
C_a	=	Total COPC air concentration (mg/m^3)
RfD	=	Reference dose (mg/kg-day)
RfC	=	Reference concentration (mg/m^3)

It should be noted that each program office within U.S. EPA determines the what *HQ* level poses a concern to exposed individuals. For example, Superfund has determined that an *HQ* of less than or equal to 1 is considered health-protective (U.S. EPA 1989e). Generally, the more that the *HQ* value exceeds 1, the greater is the level of concern. However, because *RfDs* and *RfCs* do not have equal accuracy or precision, and are not based on the same severity of effect, the level of concern does not increase linearly as an *HQ* approaches and exceeds 1 (U.S. EPA 1989e). It should also be noted that background exposures may be an important consideration in setting safe levels. This is because non-cancer effects

are generally modeled as thresholds. In specific cases, a permitting authority may elect to adjust the *HQ* downward to account for any exposure that individuals may have from other sources.

As with carcinogenic chemicals in a specific exposure pathway, a receptor may be exposed to multiple chemicals associated with noncarcinogenic health effects. The total noncarcinogenic hazard for each exposure pathway is calculated by following the procedures outlined in U.S. EPA (1986e) and U.S. EPA (1989e). Specifically, the total noncarcinogenic hazard attributable to exposure to all COPCs through a single exposure pathway is known as a hazard index (*HI*). Consistent with the procedure for addressing carcinogenic risks, the noncarcinogenic hazards from all RCRA regulated combustion units that are permitted, have interim status, or are expected to be constructed, should be summed for each receptor. Also, noncarcinogenic hazard from fugitive emissions sources, should also be included in the calculation of the *HI* for each receptor. The *HI* is calculated as follows:

$$HI = \sum_i HQ_i \qquad \text{Equation 7-5}$$

where

HI	=	Total hazard for a specific exposure pathway
HQ_i	=	Hazard quotient for COPC *i*

This summation methodology assumes that the health effects, of the various COPCs to which a receptor is exposed, are additive. Specifically, this methodology is a simplification of the *HI* concept because it does not directly consider the portal of entry associated with each exposure pathway or the often unique toxic endpoints and toxicity mechanisms of the various COPCs.

As discussed in Section 7.2 for carcinogenic risks, a receptor may be exposed to COPCs associated with noncarcinogenic health effects through more than one exposure pathway. For the purposes of the risk assessment, it is reasonable to estimate a receptor's total hazard as the sum of the *HI*s for each of the exposure pathways identified in Table 4-1. Specifically, a receptor's total hazard is the sum of hazards from each individual exposure pathway, expressed as follows:

$$Total\ HI = \sum HI \qquad \text{Equation 7-6}$$

where

Total HI	=	Total hazard from multiple exposure pathways
HI	=	Total hazard for a specific exposure pathway

Consistent with U.S. EPA guidance (1989e), all total *HI*s exceeding the target hazard level are further evaluated. The total *HI* for an exposure pathway can exceed the target hazard level as a result of the presence of either (1) one or more COPCs with an *HQ* exceeding the target hazard level, or (2) the summation of several COPC-specific *HQ*s that are each less than the target hazard level. In the former case, the presence of at least one COPC-specific hazard greater than the target hazard level is interpreted as indicating the potential for noncarcinogenic health effects. In the latter case, a detailed analysis is required to determine whether the potential for noncarcinogenic health effects is accurately estimated by the total *HI*, because the toxicological effects associated with exposure to multiple chemicals, often through different exposure pathways, may not be additive; therefore, the total *HI* may overestimate the potential for noncarcinogenic health effects. To address this issue, COPC-specific hazards are summed according to major health effects and target organs or systems (U.S. EPA 1989e). It is especially important to consider any differences related to exposure route; this process is referred to as the segregation of the *HI*. Technically, segregation of the HI based only on target organs or systems is a simplification of HI. Ideally, the HI should be segregated considering also the often unique mechanisms of toxicity of the various compounds to which receptors may be exposed. However, segregating the HI based on mechanisms of toxicity is beyond a screening level or initial risk evaluation approach.

The highest segregated *HI* resulting from this process is considered. If the segregated *HI* exceeds the target hazard level, there is a potential for noncarcinogenic health effects. However, if the segregated *HI* is less than the target hazard level, the total *HI* of all COPC-specific results likely is too conservative, and noncarcinogenic health effects are not likely to result from exposure to COPCs.

Appendix A, Table A-2 identifies target organs and systems that are affected by each COPC in each exposure route. Appendix A-3 presents *RfDs* and *RfCs* for these same COPCs. If COPCs not identified in Appendix A-3 are included in the risk assessment, *RfDs* and *RfCs* for these compounds can be

obtained from the following sources, listed in the preferred order: (1) U.S. EPA IRIS (U.S. EPA 1996a), and (2) U.S. EPA HEAST (U.S. EPA 1994b).

In the assessment of noncarcinogenic risk from COPCs, U.S EPA-derived or reviewed health benchmarks (*RfD*s, *RfC*s) are recommended. However, for numerous compounds, a complete set of inhalation and oral EPA-derived health benchmarks are not available. In such cases, the health benchmarks presented in Appendix A-3 were calculated based on available U.S. EPA-derived benchmarks values. For instance, if the *oral RfD* (mg/kg/day) was available and the *RfC* (mg/m^3) was not; the *RfC* was calculated by multiplying the *RfD* by an average human inhalation rate of 20 m^3/day and dividing by the average human body weight of 70 kg. This conversion is based on a route-to-route extrapolation, which assumes that the toxicity of the given compound is equivalent over all routes of exposure.

This process does introduce uncertainty into the risk assessment. By using this method, the risk assessor must assume that the qualitative data supporting the benchmark value for a certain route also applies to the route in question. For example, if an *RfD* is available and the *RfC* is calculated from that value, the risk assessor is assuming that the toxicity seen following oral exposure will be equivalent to toxicity following inhalation exposure. This assumption could overestimate or underestimate the toxicity of the given compound following inhalation exposure.

Because of the degree of uncertainty involved in using toxicity benchmark values calculated based on route-to-route extrapolation, a qualitative assessment of the toxicity information available for the compound and exposure route should be performed. This will enable the risk assessor to make a well informed decision concerning the validity of values calculated based on route-to-route extrapolation. This qualitative assessment should also be included in the uncertainty section of the risk assessment.

If relevant information is not available from these sources, the applicant should work with the appropriate regulatory agency to contact the U.S. EPA National Center for Environmental Assessment (NCEA) office in Cincinnati, Ohio. NCEA personnel may be able to assist in developing the necessary toxicity values.

7.4 TARGET LEVELS

Target levels are risk management based and set by the regulatory authority. Target values are not a discrete indicator of observed adverse effect. If a calculated risk falls within target values, a regulatory authority may, without further investigation, conclude that a proposed action does not present an unacceptable risk. A calculated risk that exceeds these targets, however, would not, in and of itself, indicate that the proposed action is not safe or that it presents an unacceptable risk. Rather, a risk calculation that exceeds a target value triggers further careful consideration of the underlying scientific basis for the calculation.

7.5 ACUTE EXPOSURE RESULTING FROM DIRECT INHALATION

In addition to long-term chronic effects, short-term or acute effects should be considered from direct inhalation of vapor phase and particle phase COPCs. It is assumed that short-term emissions will not have a significant impact through the indirect exposure pathways (as compared to impacts from long-term emissions). Therefore, acute effects are only evaluated through the short-term (maximum 1-hour) inhalation of vapors and particulates exposure pathway of the acute risk scenario. U.S. EPA OSW recommendations for where and when to evaluate the acute risk scenario in completing a risk assessment is described in Sections 4.2 and 4.3. In order to establish acute inhalation exposure criteria (AIEC), it was necessary to identify and evaluate (1) existing guidelines for acute inhalation exposure, and (2) existing hierarchal approaches for developing acute inhalation exposure levels. Hierarchal approaches are composed of existing guidelines for acute inhalation exposure, ranked in order of applicability and technical basis, and all being protective of the general public. It should be noted that hierarchial approaches are needed because no single organization or methodology has developed acute criteria values or benchmarks for all of the potential compounds of concern.

7.5.1 Existing Hierarchal Approaches for Acute Inhalation Exposure

Existing guidelines or criteria for evaluating acute inhalation exposure have been or are being developed by several organizations in the United States including: (1) American Conference of Governmental

Industrial Hygienists (ACGIH 1996); (2) Occupational Safety and Health Administration (NIOSH 1994); (3) National Institute of Occupational Safety and Health (NIOSH 1994); (4) American Industrial Hygiene Association (AIHA 1997); (5) National Research Council Committee on Toxicology (NRC COT 1986, U.S. EPA 1987b); (6) U.S. EPA (U.S. EPA 1987b); (7) Agency for Toxic Substances and Disease Registry (ATSDR 1997); (8) California Environmental Protection Agency (CEPA 1995); (9) National Advisory Committee (NAC 1997); and (10) Department of Energy (DoE 1997b); Subcommittee on Consequence Assessment and Protective Actions (SCAPA 1997b). Acute inhalation exposure guidelines and criteria are (1) designed to protect a variety of exposure groups including occupational workers, military personnel, and the general public, (2) based on varying exposure durations up to 24 hours in length, and (3) intended to protect against a variety of toxicity endpoints ranging from discomfort or mild adverse health effects to serious, debilitating, and potentially life-threatening effects, up to and including death.

Hierarchal approaches have been developed by a variety of organizations and teams of organizations for establishing acute inhalation exposure guidelines to protect the general public. These development organizations include:

- U.S. EPA Region 10 (U.S. EPA 1996a);
- Federal Emergency Management Agency, Department of Transportation (DoT), and U.S. EPA (U.S. EPA 1993k);
- U.S. EPA Region 3 (EPA 1996b);
- Department of Defense (DoD 1996); and
- Department of Energy (DoE) (SCAPA 1997a).

The acute inhalation exposure guidelines developed by these organizations are generally a very heterogenous group, developed to protect different subpopulations against different effects and apply to various exposure durations. Therefore, the hierarchal approaches developed by the first four organizations listed above have needed to adjust the existing guidelines using safety factors (usually multiples of 10) to account for differences in exposure group, exposure duration, and toxicity endpoint, to arrive at acute inhalation exposure values applicable to the general public.

In contrast to the hierarchal approaches developed using safety factors, the DoE's Emergency Management Advisory Committee's Subcommittee on Consequence Assessment and Protective Actions (SCAPA) has developed temporary emergency exposure limits (TEELs) based on statistical analyses between existing guidelines for acute inhalation exposure and AIHA emergency response planning guidelines (ERPG) (Craig et al. 1995). For compounds for which TEEL values could not be developed using this approach, SCAPA developed a supplementary approach using available toxicity information, primarily (1) lethal dose and concentration median, and (2) lethal dose and concentration low values (DoE 1997a).

7.5.2 U.S. EPA OSW Recommended Hierarchal Approach

After reviewing the existing hierarchal approaches, U.S. EPA OSW recommends the following approach. This approach is based on existing acute inhalation values that do not require the use of arbitrary safety factors and are intended to protect the general public from discomfort or mild adverse health effects over 1-hour exposure periods. It includes level 1 acute inhalation exposure guidelines (AEGL-1), level 1 emergency response planning guidelines (ERPG-1), and level 1 acute toxicity exposure levels (ATEL-1); supplemented with DoE TEELs and the SCAPA toxicity-based approach. The hierarchal approach is summarized below:

1. AEGL-1 (NAC 1997)
2. ERPG-1 (AIHA 1996; SCAPA 1997b)
3. ATEL-1 (Cal/EPA 1995)
4. TEEL-1 (SCAPA 1997a)
5. SCAPA Toxicity-based approach (DoE 1997a)

The hierarchy is presented in order of preference; from 1 (most preferred) to 5 (least preferred). This preference is based on (1) applicability to a 1-hour exposure duration for protection of the general public (versus only occupational exsposure), and (2) level of documentation and associated review. It should

also be noted that the hierarchy approach of preference for AEGL-1 and ERPG-1 values is also consistent with the CAAA 112r Final Rule, Risk Management Program.

To obtain a COPC-specific AIEC, one should begin with review of the AEGL-1 values specific to the COPC of interest. AEGL-1 values are currently available for 12 compounds. The Federal Register (October 30, 1997) provides a list of the proposed AEGL-1 values, which can be accessed through the web (www.EPA.FEDRGSTR). If there is not an available AEGL-1 value for a respective COPC, review the ERPG-1 values, and so forth until an AIEC value is obtained specific to the COPC of interest. Appendix A-4 provides an abbreviated listing of example AIECs developed based on values currently available for the hierarchal approach presented above.

It should also be noted that DoE's approach for developing TEELs contains existing acute inhalation exposure values that were not specifically developed to protect the general public from discomfort or mild adverse health effect over 1-hour exposure periods. AEGL-1, ERPG-1, and ATEL-1 values are all developed in accordance with the principals outlined in NRC's Committee on Toxicology (COT) guidelines for developing a detailed step-by-step process for developing defensible acute exposure levels. However, because AEGL, ERPG, and ATEL values are only available for a limited number of compounds, it becomes necessary to use TEEL values (currently available for 471 compounds).

For any COPCs for which acute inhalation exposure values cannot be developed using DoE's TEEL approach, AIECs can be developed following the toxicity-based approach used by SCAPA (Tier 5).

To characterize the potential for adverse health effects from acute exposure to COPC-specific emissions, the acute air concentration (C_{acute}) resulting from maximum emissions over a 1-hour period should be compared to the COPC-specific AIEC to calculate the acute hazard quotient (AHQ_{inh}) (see Appendix C, Table C-4-1). See Chapter 3 for discussion on air dispersion modeling related to obtaining 1-hour maximum values to calculate C_{acute} (see Appendix B, Table B-6-1). The AHQ_{inh} can be calculated as follows:

$$AHQ_{inh} = \frac{C_{acute} \cdot 0.001}{AIEC} \qquad \text{Equation 7-7}$$

where

AHQ_{inh}	=	Acute hazard quotient (unitless)
C_{acute}	=	Acute air concentration ($\mu g/m^3$)
AIEC	=	Acute inhalation exposure criteria (mg/m^3)
0.001	=	Conversion factor ($mg/\mu g$)

Acute hazard quotients should be calculated at the selected acute exposure scenario locations (see Sections 4.2 and 4.3) for COPCs specific to emissions from each source and from all facility sources combined. Target levels for acute hazard quotient evaluation is a risk management decision and will be set by the permitting authority.